AMENDMENTS TO THE SPECIFICATION:

Please amend the paragraph at page 1, line 1 to read as follows:

This is a continuation of Application No. 09/534,072, filed March 24, 2000 (~~allowed~~ issued on December 7, 2004, as U.S. Patent No. 6,828,429), ~~and~~, which claims the benefit of U.S. Provisional Application No. 60/126,469, filed March 26, 1999, all of which are incorporated herein by reference.

Please replace the paragraph spanning pages 16-17 with the following paragraph, amended to remove embedded hyperlinks. The text in the third line of the paragraph was underlined in the original application and that underlining does not represent an addition to the text.

For example, chromosomes can be mapped by radiation hybridization. First, PCR is performed using the Whitehead Institute/MIT Center for Genome Research Genebridge4 panel of 93 radiation hybrids ~~(http://www-genome.wi.mit.edu/ftp/distribution/human_STS_releases/july97/rhmap/genebridge4.html)~~. Primers are used which lie within a putative exon of the gene of interest and which amplify a product from human genomic DNA, but do not amplify hamster genomic DNA. The results of the PCRs are converted into a data vector that is submitted to the Whitehead/MIT Radiation Mapping site on the internet ~~(http://www-seq.wi. mit.edu)~~. The data is scored and the chromosomal assignment and placement relative to known Sequence Tag Site (STS) markers on the radiation hybrid map is provided. ~~(The following web site provides additional information about radiation hybrid mapping: http://www-genome.wi.mit.edu/ftp/distribution/human_STS_releases/july97/07-97.INTRO.html).~~

Please replace the paragraph spanning pages 20-21 with the following paragraph, amended to demarcate a trademark.

Genomic DNA-free total RNA was extracted from this enriched pool of cells using RNA[[zol]]ZOL™ B (Tel-Test, Inc., Friendswood, TX) according to manufacturer's protocol. The epithelial nature of the RNA source was further confirmed using cytokeratin 18 expression (45) in reverse transcriptase-polymerase chain reaction (RT-PCR) assays. Using arbitrary and anchored primers containing 5' M13 or T7 sequences (obtained from Biomedical Instrumentation Center, Uniformed Services University of the Health Sciences, Bethesda), the isolated DNA-free total RNA was amplified by RT-PCR which was performed using ten anchored antisense primers and four arbitrary sense primers according to the protocol provided by Hieroglyph™ RNA Profile Kit 1 (Genomyx Corporation, CA). The cDNA fragments produced by the RT-PCR assay were analyzed by high resolution gel electrophoresis, carried out by using Genomyx™ LR DNA sequencer and LR-Optimized™ HR-1000™ gel formulations (Genomyx Corporation, CA).

Please replace the paragraph on page 21, beginning on line 10 with the following paragraph, amended to demarcate a trademark.

All the reamplified cDNA fragments were purified by ~~Centricon~~CENTRICON™-c-100 system (Amicon, USA). The purified fragments were sequenced by cycle sequencing and DNA sequence determination using an ABI 377 DNA sequencer. Isolated sequences were analyzed for sequence homology with known sequences by running searches through publicly available DNA sequence databases, including the National Center for Biotechnology Information and the Cancer Genome Anatomy Project. Approximately two-thirds of these cDNA sequences exhibited homology to previously described DNA sequences/genes e.g., ribosomal proteins, mitochondrial DNA sequences, growth factor receptors, and genes involved in maintaining the redox state in cells. About one-third of the cDNAs represented novel sequences, which did not exhibit similarity to the sequences available in publicly available databases. The PCGEM1

fragment, obtained from the initial differential display screening represents a 530 base pair (nucleotides 410 to 940 of SEQ ID NO: 1) cDNA sequence which, in initial searches, did not exhibit any significant homology with sequences in the publicly available databases. Later searching of the high throughput genome sequence (HTGS) database revealed perfect homology to a chromosome 2 derived uncharacterized, unfinished genomic sequence (accession # AC 013401).

Please replace the paragraph spanning pages 26-27 with the following paragraph, amended to demarcate a trademark.

LNCaP cells were maintained in RPMI 1640 (Life Technologies, Inc., Gaithersburg, MD) supplemented with 10% fetal bovine serum (FBS, Life Technologies, Inc., Gaithersburg, MD) and experiments were performed on cells between passages 20 and 35. For the studies of NKX3.1 gene expression regulation, charcoal/dextran stripped androgen-free FBS (cFBS, Gemini Bio-Products, Inc., Calabasas, CA) was used. LNCaP cells were cultured first in RPMI 1640 with 10% cFBS for 4 days and then stimulated with a non-metabolizable androgen analog R1881 (DUPONT, Boston, MA) at different concentrations for different times as shown in Figure 5A. LNCaP cells identically treated but without R1881 served as control. Poly A+ RNA derived from cells treated with/without R1881 was extracted at indicated time points with RNA[[zol]]ZOL™ B (Tel-Test, Inc, TX) and fractionated (2 µg/lane) by running on 1% formaldehyde-agarose gel and transferred to nylon membrane. Northern blots were analyzed for the expression of PCGEM1 using the nucleic acid molecule set forth in SEQ ID NO: 4 as a probe. The RNA from LNCaP cells treated with R1881 and RNA from control LNCaP cells were also analyzed by RT-PCR assays as described in Example 4.

Please replace the first full paragraph on page 29 with the following paragraph, amended to remove an embedded hyperlink and correct a typographical error.

The Codon Preference program (GCG Wisconsin Package, Madison, WI), which locates protein coding regions in a reading frame specific manner further suggested the absence of protein coding capacity in the PCGEM1 gene ~~(see www.cpdr.org)~~. *In vitro* transcription/translation of PCGEM1 cDNA did not produce a detectable protein/peptide. Although we can not unequivocally rule out the possibility that PCGEM1 codes for a short unstable peptide, at this time both experimental and computational approaches strongly suggest that PCGEM1 cDNA does not have protein coding capacity. (It should be recognized that conclusions regarding the role of PCGEM1 are speculative in nature, and should not be considered limiting in any way).